Letters

High Prevalence of Sin Nombre Virus in Rodent Populations, Central Utah: A Consequence of Human Disturbance?

To the Editor: Sin Nombre virus (SNV) (Bunyaviridae) is a newly discovered hantavirus responsible for hantavirus pulmonary syndrome (HPS) in humans. The deer mouse, *Peromyscus maniculatus,* is its primary reservoir (1,2). To address a gap in our understanding of the temporal dynamics of SNV, we conducted a longitudinal study in the Great Basin.

Our study site was the West Tintic Mountains, Jericho, Utah, 39°57' N, 112°22' W. We trapped on May 29 to 31, July 10 to 12, and October 7 to 9, 1999. Previous research (3) indicated woodrats (*Neotoma lepida*) were reservoirs for SNV; therefore, we concentrated our trapping efforts at woodrat middens. Middens (2 m diam) are structures of thousands of sticks built by woodrats and serve as nesting sites for a variety of small rodents, including deer mice (4,5). Each night of the 3-night trapping session, we set ~3 live traps baited with oats, peanut butter, and cotton at each of ~40 middens.

Captured rodents were collected each morning and anesthetized with Metafane (methoxyflurane). Animals were identified to species and ear-clipped for future identification. Scarring, body mass, sex, tail length, and reproductive status were recorded (data available by request). Animals were bled via the retroorbital sinus. We performed an enzyme-linked immunosorbent assay for detection of hantavirus antibody (3).

We trapped six species of rodents; *P. maniculatus* was the most common, followed by *P. truei* and *N. lepida*. Other species captured infrequently were *Dipodomys ordii*, *Largurus curtatus*, and *Chaetodipus* sp.

Over three trapping periods, we captured 212 *P. maniculatus;* 63 were antibody-positive (29.7%). Prevalence of SNV was greater in males than in females (chi-square = 3.8, p = 0.05), and it varied little among sampling periods. Most of the variation was due to changes in prevalence in males, which was 28% to 42%; prevalence among female deer mice was 17% to 20%. *P. truei* also tested positive for SNV. Of 37 *P. truei* tested, 4 were antibody-positive (10.8%).

We found a high and relatively stable level of SNV prevalence in a population of deer mice in Central Utah. Mean antibody prevalence (29.7%) across 3 periods was up to 3 times higher than that of other locations. Prevalence of SNV in this population was comparable with that during the 1993 outbreak of HPS in the Four Corners region.

We propose that the high level of SNV prevalence could be due to disturbance by humans, primarily intensive use of all-terrain vehicles at the study site. Little Sahara Recreation Area, ~4 km from the study site, recorded 180,000 visitors during 1999, mostly all-terrain vehicle users (Bureau of Land Management statistics). Many visitors to Little Sahara camp and recreate on land in our study area. This heavy recreational use has produced numerous dirt roads and campsites. Vehicle movement has denuded the area of vegetation other than large (>1 m tall) shrubs and trees and has removed cryptogamic crust, resulting in compaction of sandy soil into roads, trails, and large open

spaces. Open spaces caused by disturbance reduce habitat suitable for species such as *Peromyscus* (6,7) and may cause animal density to increase within a microhabitat. Increased intra- and interspecific interactions would favor the transmission of SNV. Thus, fragmentation of the landscape may alter behavior of deer mice in a manner that enhances transmission of SNV.

Four pieces of evidence corroborate our speculation that habitat disturbance increases prevalence of SNV. First, in experimentally fragmented habitats, the density of deer mice increased dramatically. In one study, density of deer mice in small patches (4 m x 8 m) was consistently 3 times higher during 7 years of the study than that of deer mice in larger patches (10 x 50 m) (6). These small patches are similar in size to patches created by vehicle movement at our study site. Second, deer mice in fragmented habitats travel much longer distances, on average 2 times as far, as deer mice in less fragmented habitats (6). Third, immunocompetence of small rodents may decline as population density increases, making rodents more susceptible to infection than at lower densities (8). These three factors taken together should enhance transmission of SNV by increasing interactions among deer mice with lowered immunocompetence. Finally, prevalence of SNV in deer mice is lower in populations from habitats less impacted but similar to our study site. Across four other sites in the Great Basin, prevalence of SNV was 11% (9). Although we have not quantified the disturbance in these other areas, their general locations suggest they are not as disturbed by humans as the site near Little Sahara Recreational Area.

Further investigation of the effect of human disturbance on SNV prevalence is needed. We have presented several possible mechanisms that may be involved in a causal relationship between these two factors. Given that most HPS cases are contracted in areas where there has been human alteration to the landscape, future investigation of this hypothesis is warranted.

The prevalence and total numbers of infected rodents were much lower in *P. truei* than *P. maniculatus*. Adult *P. truei* are larger than *P. maniculatus* and tend to compete with *P. maniculatus* for food and nesting sites. Interspecific competition could lead to aggressive contact between these two species that could result in interspecific transmission of SNV. *P. truei* were regularly captured at the same middens on the same nights with *P. maniculatus*.

We suggest that the high level of disturbance at this study site could increase the probability of SNV transmission between species through the same mechanisms suggested for the high levels of prevalence within deer mice. Rodents at our study site may be living at higher densities than in other areas. The increased contact between species, especially when SNV prevalence is high in deer mice, could promote transmission to species other than *P. maniculatus*.

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